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(54) System and method for prevention of recurrent vasovagal syncope using cardiac pacing

(57) A pacemaker or other cardiac stimulation device (10) is configured to predict the onset of vasovagal syncope and administers pacing therapy to prevent the syncope from occurring. Prediction of the onset of vasovagal syncope is based upon an analysis of the contractility of the heart muscle. In an example described herein, the contractility of the heart muscle is detected and compared with the average contractility. If the current contractility exceeds the average contractility by a predetermined threshold, a high risk of vasovagal syncope is thereby detected and the heart is paced at a vasovagal syncope prevention rate which may be, for example, 20 to 40 beats per minute faster than a previous heart rate. The contractility of the heart is determined, for example, by measuring the impedance of the heart tissue or by measuring the movement of heart tissue in the wall of the heart or by pressure using an accelerometer. The contractility is averaged over a period of time to determine the average contractility.

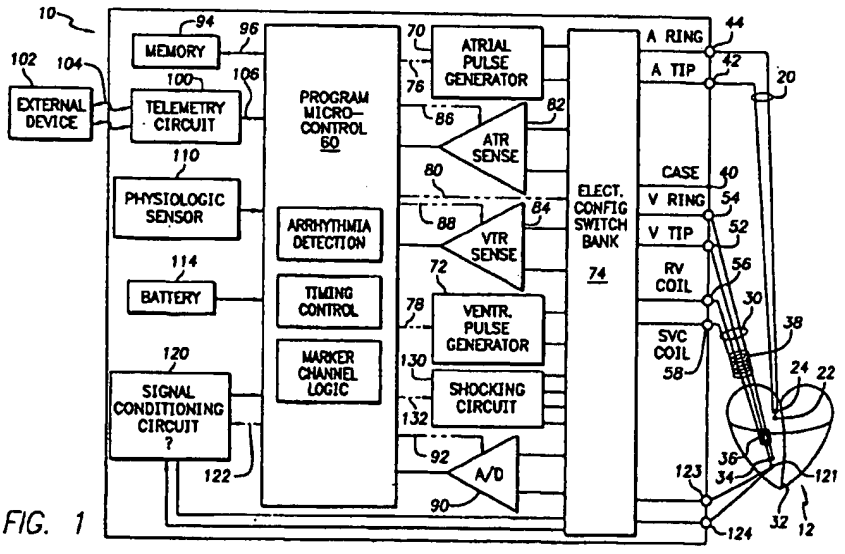

FIG. 1

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Description

[0001] The invention generally relates to cardiac pacing techniques and in particular to techniques for preventing vasovagal syncope using cardiac pacing.

[0002] Syncope is a sudden loss of strength or consciousness caused by reduced cerebral circulation, itself typically the result of vasodilation. Vasovagal syncope is a type of syncope referred to as a neurocardiogenic syncope wherein the syncope is triggered by an interaction between the heart and nerve tissue connected to the heart. Neurocardiogenic syncope may also be referred to as neuromediated syncope, neurally mediated syncope, neurocardiogenic syncope, cardioneurogenic syncope, vasodepressor syncope, malignant vasovagal syndrome, neurally mediated hypotension/bradycardia and cardiovascular neurogenic syncope. For vasovagal syncope, the interaction occurs between the heart and the vagus nerve.

[0003] Evidence suggests that vasovagal syncope is initially triggered by a sudden reduction in peripheral vascular resistance, perhaps resulting from stress, pooling of blood in the extremities, or other factors. As a result of the reduction in peripheral vascular resistance, the pressure of blood entering the heart drops and the amount of blood filling the ventricles prior to ventricular contractions therefore also drops. With less blood in the ventricles, the ventricles thereby contract much more quickly and vigorously than would otherwise occur in an effort to maintain a constant stroke volume. The more vigorous ventricular contractions have the effect of stimulating ventricular mechanoreceptors, also known as C fibers, that normally only respond to ventricular expansion or stretching, rather than contraction. The activation of the ventricular mechanoreceptors results in a surge in neural traffic to the brainstem, particularly to the nucleus tractus solitaries, via the vasovagal nerve.

[0004] For most people, the neurological system properly interprets the increase of activity of the mechanoreceptors as being in response to a drop in peripheral vascular resistance and compensates by increasing the heart rate and constricting the blood vessels. However, in certain patients, as a result of a neurological condition within the vagus nerve or for some other reason, the surge in neural traffic is falsely perceived by the neurological system as being representative of hypertension. In response thereto, the brainstem triggers an increase in peripheral vasodilation and a reduction in heart rate. The vasodilation and the drop in heart rate, in turn, cause a still further reduction in blood pressure, i.e. hypotension. In other words, the actions taken by the brainstem exacerbate the problem. If the degree of hypotension is sufficiently severe, cerebral hypoperfusion occurs wherein brain cells do not receive enough oxygen and, consequently, the victim loses consciousness. Accordingly, within these patients, any sudden drop in peripheral vascular resistance can trigger vasovagal syncope and the patients are the to suffer from recurrent vasovagal syncope. Further information regarding vasovagal syncope may be found in S. Serge Harold and Jacques Mugica, *Recent Advances in Cardiac Pacing,* Futura Publishing Company, 92-95, 1997.

[0005] As can be appreciated, loss of consciousness can be particularly dangerous for the patient if occurring while the patient is driving a motor vehicle, climbing a flight of stairs or engaged in any other activity wherein the loss of consciousness could result in injury or death. Accordingly, it is highly desirable to provide techniques for preventing vasovagal syncope or other forms of neurocardiological syncope. One possible technique for preventing vasovagal syncope is to employ a pacemaker, or other implantable cardiac pacing device, to pace the heart to prevent the reduction in blood pressure associated with vasovagal syncope from occurring. Indeed, the American College of Cardiology-American Heart Association suggested in 1991 that vasovagal syncope in patients should be used as a Class 2 indication for pacing therapy. However, conventional cardiac pacemakers have had only limited success in preventing recurrent vasovagal syncope. (See David G. Benditt et al., *Cardiac Pacing for Prevention of Recurrent Vasovagal Syncope,* Ann Intern Med. 1995; 122; 204-209.) Insofar as vasovagal syncope is concerned, conventional pacemakers only respond to the reduction in heart rate associated therewith. The reduction in heart rate, also referred to as bradycardia, is detected by the pacemaker, which increases the heart rate to compensate Depending upon the programming of the pacemaker, it may take five to eight seconds or more before the pacemaker begins increasing the heart rate in response to detection of bradycardia. For many patients subject to recurrent vasovagal syncope, a significant drop in blood pressure occurs before there is any bradycardia. Hence, by the time the pacemaker begins to increase the heart rate, the blood pressure will already have dropped to the point where the increase in heart rate is ineffective. In this regard, the drop in blood pressure results in significantly less blood filling the ventricles, such that there is simply not enough incoming blood to pump. Hence, overall blood pressure is not significantly increased merely by pumping the heart faster, and the aforementioned cerebral hypoperfusion still occurs resulting in unconsciousness. Moreover, for at least some patients subject to recurrent vasovagal syncope, there is no significant drop in heart rate, only a drop in blood pressure. Hence, pacemakers programmed to prevent vasovagal syncope based solely upon detection of bradycardia will have little or no effect.

[0006] Thus, bradycardia-triggered vasovagal syncope prevention techniques employed by some conventional cardiac pacemakers often fail to prevent the syncope. Moreover, conventional cardiac pacemakers are often unable to distinguish physiologic bradycardia such as that associated with sleep from bradycardia associated with vasovagal syncope or other neurocardiogenic syncope.

[0007] Accordingly, it would be highly desirable to provide improved cardiac pacing devices capable of predicting the onset of vasovagal syncope or other types of neurocardiogenic syncope, and for reliably administering pacing therapy to prevent the syncope from occurring. It is to these ends that the invention is primarily directed.

[0008] In accordance with one aspect of the invention, a cardiac pacing device is provided with a vasovagal syncope prediction and prevention unit for predicting the onset of vasovagal syncope and for administering pacing therapy to prevent the syncope from occurring. Prediction of the onset of vasovagal syncope is based upon an analysis of the contractility of the heart muscle.

[0009] In an exemplary embodiment, prediction of the onset of vasovagal syncope is achieved by sensing the contractility of the heart muscle and determining whether the sensed contractility exceeds an average contractility by a predetermined threshold amount. If so, the heart is paced at a vasovagal syncope prevention rate which may be, for example, 20 to 40 beats per minute faster than a previous heart rate. The contractility of the heart is determined, for example, by measuring the electrical impedance change or pressure change in the ventricle, or by measuring the movement of heart tissue in the wall of the heart using an accelerometer. The contractility measured is averaged over a period of time to determine the average contractility.

[0010] In this manner, the onset of vasovagal syncope is predicted based upon changes in heart muscle contractility and syncope is substantially prevented from occurring by immediately administering vasovagal syncope preventative pacing measures. Because prediction of imminent vasovagal syncope is based upon a measurement of the contractility of the heart tissue, rather than upon detection of an episode of bradycardia, vasovagal syncope is thereby detected even in circumstances wherein hypotension occurs before bradycardia or wherein bradycardia does not occur at all. Also, physiologic episodes of bradycardia, such as those associated with sleep, do not erroneously trigger the vasovagal syncope pacing therapy. Moreover, the increase in contractility associated with vasovagal syncope can be detected quickly whereas reliable bradycardia detection may require analysis of several heart beats. Hence, preventative pacing may be administered before the blood pressure has dropped significantly, as could otherwise occur if relying solely on bradycardia detection. Two or more independent contractility sensors may be provided to help prevent a false positive detection of vasovagal syncope.

[0011] Method embodiments of the invention are also provided as set out in the appended claims.

[0012] In a preferred aspect of the method, the step of detecting the contractility of the heart is performed to detect contractility of the left ventricle.

[0013] Preferably the step of detecting the contractility of the heart includes the step of detecting the electrical impedance of blood within one of the ventricles of the heart.

[0014] Preferably the step of detecting the contractility of the heart includes the step of detecting the movement of muscle tissue in the wall of one of the ventricles of the heart using an accelerometer.

[0015] Preferably the step of determining whether there is a significant risk of the onset of vasovagal syncope includes the steps of:

determining an average contractility of the heart for a plurality of cardiac cycles;

comparing the contractility of the heart detected during a current cardiac cycle with an average contractility of the heart a plurality of cardiac cycles; and

concluding that there is a significant risk of the onset of vasovagal syncope if the contractility of the heart detected during the current cardiac cycle exceeds the average contractility of the heart for a plurality of cardiac cycles by a predetermined threshold, and preferably the step of determining the average contractility of the heart for a plurality of cardiac cycles includes the step of iteratively updating the average contractility using each new value of the detected contractility.

[0016] Preferably the implantable medical device is a cardiac pacing device and wherein the method further includes the step of:

responsive to a determination that there is a significant risk of the onset of vasovagal syncope, pacing the heart to reduce the likelihood of the vasovagal syncope and preferably the step of pacing the heart to reduce the likelihood of the vasovagal syncope includes the steps of:

incrementally increasing a pacing rate from a preexisting pacing rate to a predetermined vasovagal syncope prevention rate during a first predetermined period of time;

maintaining the pacing rate at the predetermined vasovagal syncope prevention rate during a second predetermined period of time; and

incrementally decreasing the pacing rate from the vasovagal prevention rate to the preexisting pacing rate during a third predetermined period of time, and preferably the first, second and third predetermined period of times are in the range of 3 to 5 seconds, 30 to 180 seconds, and 10 to 30 seconds, respectively, and/or the vasovagal syncope prevention rate is in the range of 20 to 40 beats per minute above the preexisting pacing rate, and or if the preexisting pacing rate exceeds the vasovagal syncope prevention rate, the pacemaker continues to pace at the preexisting rate, and/or the preexisting pacing rate is a predetermined base pacing rate, and/or the preexisting pacing rate is a sensor responsive pacing rate.

[0017] Embodiments of the invention will now be described, by way of example, with reference to the drawings of which:

Fig. 1 is a functional block diagram of a dual-chamber implantable stimulation device illustrating the basic elements of a stimulation device which can provide cardioversion, defibrillation and pacing stimulation and can predict and prevent vasovagal syncope in accordance with an exemplary embodiment of the invention;

FIG. 2 is a graph illustrating a vasovagal syncope preventative pacing regime;

FIG. 3 is a flow chart illustrating steps performed by the pacemaker components of FIG. 2; and

FIGS. 4-5 illustrate a detailed flow chart describing the steps performed by a pacemaker configured to pace the heart using a sensor indicated rate (SIR) and also configured to perform vasovagal syncope preventative pacing.

[0018] In the description of the invention that follows, like numerals or reference designators will be used to refer to like parts or elements throughout.

[0019] In Fig. 1, a simplified block diagram is shown of a dual-chamber implantable stimulation device 10 which is capable of treating both fast and slow arrhythmias with stimulation therapy, including cardioversion, defibrillation, and pacing stimulation. While a dual-chamber device is shown, this is for illustration purposes only, and one of skill in the art could readily eliminate or disable the appropriate circuitry to provide a single-chamber stimulation device capable of treating one chamber with cardioversion, defibrillation and pacing stimulation.

[0020] To provide atrial chamber pacing stimulation and sensing, the stimulation device 10 is shown in electrical communication with a patient's heart 12 by way of an implantable atrial lead 20 having an atrial tip electrode 22 and an atrial ring electrode 24 which typically is implanted in the patient's atrial appendage.

[0021] The stimulation device 10 is also shown in electrical communication with the patient's heart 12 by way of an implantable ventricular lead 30 having, in this embodiment, a ventricular tip electrode 32, a ventricular ring electrode 34, a right ventricular (RV) coil electrode 36, and an SVC coil electrode 38. Typically, the ventricular lead 30 is transvenously inserted into the heart 12 so as to place the RV coil electrode 36 in the right ventricular apex, and the SVC coil electrode 38 in the superior vena cava. Accordingly, the ventricular lead 30 is capable of receiving cardiac signals, and delivering stimulation in the form of pacing and shock therapy to the right ventricle.

[0022] While only two leads are shown in FIG. 1, it is to be understood that additional stimulation leads (with one or more pacing, sensing and/or shocking electrodes) may be used in order to efficiently and effectively provide pacing stimulation to the left side of the heart or atrial cardioversion and/or defibrillation. For example, a lead designed for placement in the coronary sinus region could be implanted to deliver left atrial pacing, atrial shocking therapy, and/or for left ventricular pacing stimulation. For a complete description of a coronary sinus lead, see U.S. Patent Application No. 09/196,898, "A Self-Anchoring Coronary Sinus Lead" (Pianca et. al), and U.S. Patent No. 5,466,254, "Coronary Sinus Lead with Atrial Sensing Capability" (Helland), which patents are hereby incorporated herein by reference.

[0023] The housing 40 (shown schematically) for the stimulation device 10 includes a connector (not shown) having an atrial pin terminal 42 and an atrial ring terminal 44, which are adapted for connection to the atrial tip electrode 22 and the atrial ring electrode 24, respectively. The housing 40 further includes a ventricular pin terminal 52, a ventricular ring terminal 54, a ventricular shocking terminal 56, and an SVC shocking terminal 58, which are adapted for connection to the ventricular tip electrode 32, the ventricular ring electrode 34, the RV coil electrode 36, and the SVC coil electrode 38, respectively. The housing 40 (often referred to as the "can", "case" or "case electrode") may be programmably selected to act as the return electrode, or anode, alone or in combination with one of the coil electrodes, 36 and 38. For convenience, the names of the electrodes are shown next to the terminals.

[0024] At the core of the stimulation device 10 is a programmable microcontroller 60 which controls the various modes of stimulation therapy. As is well known in the art, the microcontroller 60 includes a microprocessor, or equivalent control circuitry, designed specifically for controlling the delivery of stimulation therapy and may further include RAM or ROM memory, logic and timing circuitry, state machine circuitry, and I/O circuitry. Typically, the microcontroller 60 includes the ability to process or monitor input signals (data) as controlled by a program code stored in a designated block of memory. The details of the design and operation of the microcontroller 60 are not critical to the present invention. Rather, any suitable microcontroller 60 may be used that carries out the functions described herein. The use of microprocessor-based control circuits for performing timing and data analysis functions is well known in the art. As shown in Fig. 1, an atrial pulse generator 70 and a ventricular pulse generator 72 generate pacing stimulation pulses for delivery by the atrial lead 20 and the ventricular lead 30, respectively, via a switch bank 74. The pulse generators, 70 and 72, are controlled by the microcontroller 60 via appropriate control signals, 76 and 78, respectively, to trigger or inhibit the stimulation pulses. The microcontroller 60 further includes timing circuitry that controls the operation of the stimulation device timing of such stimulation pulses , that is well known in the art.

[0025] The switch bank 74 includes a plurality of switches for switchably connecting the desired elec-

trodes to the appropriate I/O circuits, thereby providing complete electrode programmability. Accordingly, the switch bank 74, in response to a control signal 80 from the microcontroller 60, determines the polarity of the stimulation pulses (e.g., unipolar or bipolar) by selectively closing the appropriate combination of switches (not shown) as is known in the art. An atrial sense amplifier 82 and a ventricular sense amplifier 84 are also coupled to the atrial and ventricular leads 20 and 30, respectively, through the switch bank 74 for detecting the presence of cardiac activity. The switch bank 74 determines the "sensing polarity" of the cardiac signal by selectively closing the appropriate switches, as is also known in the art. In this way, the clinician may program the sensing polarity independent of the stimulation polarity.

[0026] Each sense amplifier, 82 and 84, preferably employs a low power, precision amplifier with programmable gain and/or automatic gain control, bandpass filtering, and a threshold detection circuit, known in the art, to selectively sense the cardiac signal of interest. The automatic gain control enables the device 10 to deal effectively with the difficult problem of sensing the low frequency, low amplitude signal characteristics of ventricular fibrillation.

[0027] The outputs of the atrial and ventricular sense amplifiers, 82 and 84, are connected to the microcontroller 60 which, in turn, inhibit the atrial and ventricular pulse generators, 70 and 72, respectively, in a demand fashion whenever cardiac activity is sensed in the respective chambers.

[0028] For arrhythmia detection, the present device utilizes the atrial and ventricular sense amplifiers, 82 and 84, to sense cardiac signals to determine whether a rhythm is physiologic or pathologic. As used herein "sensing" is reserved for the noting of an electrical depolarization, and "detection" is the processing of these sensed depolarization signals and noting the presence of an arrhythmia. The timing intervals between sensed events (e.g., the P-P and R-R intervals) are then classified by the microcontroller 60 by comparing them to a predefined rate zone limit (i.e., bradycardia, normal, low rate VT, high rate VT, and fibrillation rate zones) and various other characteristics (e.g., sudden onset, stability, physiologic sensors, and morphology, etc.) in order to determine the type of remedial therapy that is needed (e.g., bradycardia pacing, anti-tachycardia pacing, cardioversion shocks or defibrillation shocks, also known as "tiered therapy").

[0029] Cardiac signals are also applied to the inputs of an analog to digital (A/D) data acquisition system 90. The data acquisition system 90 is configured to acquire intracardiac electrogram signals, convert the raw analog data into a digital signal, and store the digital signals for later processing and/or telemetric transmission to an external device 102. The data acquisition system 90 is coupled to the atrial and ventricular leads, 20 and 30, through the switch bank 74 to sample cardiac signals across any pair of desired electrodes.

[0030] The microcontroller 60 is further coupled to a memory 94 by a suitable data/address bus 96, wherein the programmable operating parameters used by the microcontroller 60 are stored and modified, as required, in order to customize the operation of the stimulation device 10 to suit the needs of a particular patient. Such operating parameters define, for example, pacing pulse amplitude, pulse duration, electrode polarity, rate, sensitivity, automatic features, arrhythmia detection criteria, and the amplitude, waveshape and vector of each shocking pulse to be delivered to the patient's heart 28 within each respective tier of therapy.

[0031] Advantageously, the operating parameters of the implantable device 10 may be non-invasively programmed into the memory 94 through a telemetry circuit 100 in telemetric communication with an external device 102, such as a programmer, transtelephonic transceiver, or a diagnostic system analyzer. The telemetry circuit 100 is activated by the microcontroller by a control signal 106. The telemetry circuit 100 advantageously allows intracardiac electrograms and status information relating to the operation of the device 10 (as contained in the microcontroller 60 or memory 94) to be sent to the external device 102 through the established communication link 104.

[0032] In the preferred embodiment, the stimulation device 10 further includes a physiologic sensor 110. Such sensors are commonly called "rate-responsive" sensors. The physiological sensor 110 is used to detect the exercise state of the patient, to which the microcontroller 60 responds by adjusting the rate and AV Delay at which the atrial and ventricular pulse generators, 70 and 72, generate stimulation pulses. The type of sensor used is not critical to the present invention and is shown only for completeness.

[0033] The stimulation device additionally includes a battery 114 which provides operating power to all of the circuits shown in **Fig. 1**. For the stimulation device 10, which employs shocking therapy, the battery must be capable of operating at low current drains for long periods of time , and then be capable of providing high-current pulses (for capacitor charging) when the patient requires a shock pulse. The battery 114 must also have a predictable discharge characteristic so that elective replacement time can be detected. Accordingly, the device employs lithium/silver vanadium oxide batteries, as is true for most (if not all) such devices to date.

[0034] It is the primary function of the invention to function as an implantable cardioverter/defibrillator (ICD) device. That is, it must detect the occurrence of an arrhythmia, and automatically apply an appropriate electrical shock therapy to the heart aimed at terminating the detected arrhythmia. To this end, the microcontroller 60 further controls a shocking circuit 130 by way of a control signal 132. The shocking circuit 130 generates shocking pulses of low (up to 0.5 Joules), moderate (0.5 - 10 Joules), or high energy (11 to 40 Joules), as

controlled by the microcontroller 60. Such shocking pulses are applied to the patient's heart through at least two shocking electrodes, and as shown in this embodiment, using the RV and SVC coil electrodes, 36 and 38, respectively. In alternative embodiments, the housing 40 may act as an active electrode in combination with the RV electrode 36 alone, or as part of a split electrical vector using the SVC coil electrode 38 (i.e., using the RV electrode as common).

[0035] Cardioversion shocks are generally considered to be of low to moderate energy level (so as to minimize pain felt by the patient), and/or synchronized with an R-wave and/or pertaining to the treatment of tachycardia. Defibrillation shocks are generally of moderate to high energy level (i.e., corresponding to thresholds in the range of 5-40 Joules), delivered asychronously (since R-waves may be too disorganized), and pertaining exclusively to the treatment of fibrillation. Accordingly, the microcontroller 60 is capable of controlling the synchronous or asynchronous delivery of the shocking pulses.

[0036] In addition to the aforementioned components, ventricular lead 30 includes at least one contractility sensor 121, e.g., an accelerometer, a pressure sensor, and/or an impedance sensor (hereinafter referred to as the "contractility sensor(s) 121"). Device 10 is configured operate to sense the contractility of the heart using the contractility sensor(s) 121 on ventricular lead 20 and, in response to a sudden increase in the contractility of the heart, to immediately pace the heart at an elevated rate to prevent vasovagal syncope from occurring, or to delay the progression of vasovagal syncope. To this end, device 10 includes signal conditioning circuitry 120 or other input circuit which receives analog signals via one or more terminals 123 and 124 from the contractility sensor(s) and converts the analog signals into digital signals from processing by controller 60. Controller 60 includes software for analyzing the signals received from the conditioning circuitry to determine the contractility of the heart. More specifically, controller 60 analyzes the signals to first estimate the amount of blood within the right ventricle. Based upon the estimate of the amount of blood in the right ventricle, controller 60 further then estimates the contractility of the muscles of the right ventricle.

[0037] The controller also determines the average contractility and stores it in memory 94 or within a separate contractility storage register (not shown). Controller 60 then compares the sensed contractility of the heart with the average contractility of the heart and controls atrial and ventricular pulse generators 70 and 72 to administer pacing pulses to the heart at an accelerated rate in an effort to compensate for the reduction in blood pressure otherwise occurring prior to vasovagal syncope.

[0038] As noted, controller 60 may determine contractility based on pressure, acceleration or pressure signals, or a combination thereof, received from the contractility sensor(s). One technique for estimating contractility based upon electrical impedance detected within the heart is described within,U.S. Patent No. 5,800,467 to Park et al., entitled "Cardio-Synchronous Impedance Measurement System for an Implantable Stimulation Device", which is incorporated by reference herein. Briefly, blood is an electrical conductor and, hence, a decrease in the amount of blood in the ventricle results in an increase in electrical impedance. Impedance therefore, as measured within the ventricle of the heart, provides an indication of the amount of blood in the ventricle. Controller 60 monitors the impedance of the heart and detects any sharp increase in the impedance from one cycle to the next. The increase is impedance is therefore interpreted as a decrease in the amount of blood filling the ventricle during the cardiac cycle. As noted above, a sudden drop in ventricular filling causes the ventricle to contract more vigorously, i. e. the contractility of the ventricle increases. The sharp increase in contractility causes mechano-receptors within the ventricle to transmit signals to the vagus nerve which are forwarded to the brainstem. For patients with recurrent vasovagal syncope, the resulting surge in signals is misinterpreted as being representative of hypertension and the brainstem dilates blood vessels and decreases the heart rate in an attempt to compensate for the hypertension. This results in a significant drop in blood pressure causing hypoperfusion of the cerebrum and possibly loss of consciousness. Hence, a sharp increase in impedance from one period of ventricular contraction to the next is a good indicator of the potential onset of vasovagal syncope, at least within patients who suffer from recurrent vasovagal syncope.

[0039] One technique for estimating contractility based upon signals from an accelerometer within the right ventricle is as follows. The accelerometer generates signals representative of the speed at which the ventricular wall moves during a contraction. The increase in contractility caused by a sudden drop in ventricular filling results in the ventricular walls moving more forcefully. Hence, by monitoring the signals received from the accelerometer, controller 60 is able to estimate the contractility of the ventricle.

[0040] One specific technique for measuring heart contractility using an accelerometer is described within U.S. Patent No. 5,480,412 to Mouchawar et al., entitled "System and Method for Deriving Hemodynamic Signals from a Cardiac Wall Motion Sensor", which is incorporated by reference herein. One specific technique for measuring heart contractility using an impedance sensor is described within U.S. Patent No. 5,139,020 to Koestner, et al., entitled "Method and Apparatus for Controlling the Hemodynamic State of a Patient Based on Systolic Time Interval Measurements Detecting Using Doppler Ultrasound Techniques", which is incorporated by reference herein.

[0041] Thus, controller 60 tracks the average and current contractility of the heart. The average contractility,

stored within memory 94, is determined, for example, by averaging the measured contractility over relatively long periods of time such as minutes, hours, days, weeks, months or years. Controller 60 periodically compares the measured contractility with the average contractility and detects any sharp increase in contractility from one cardiac cycle to the next. As noted above, a sharp increase in contractility from one period to the next is indicative of the onset of vasovagal syncope, at least within patients subject to recurrent vasovagal syncope syndrome. If a sharp increase in contractility is detected, the controller promptly controls atrial and ventricular pulse generators 70 and 72 to increases the heart rate to compensate for the drop in blood pressure that initially triggered the increase in contractility.

[0042] In **Fig. 2**, a flow chart is shown describing an overview of the operation of the novel features of the present invention. In this flow chart, and the other flow charts described herein, the various algorithmic steps are summarized in individual "blocks". Such blocks describe specific actions or decisions made or carried out as the algorithm proceeds. Where a microcontroller (or equivalent) is employed, the flow charts presented herein provide the basis for a "control program" that may be used by such a microcontroller (or equivalent) to effectuate the desired control of the stimulation device. Those skilled in the art may readily write such a control program based on the flow charts and other descriptions presented herein.

[0043] More specifically, **FIG. 2** illustrates a pacing regime which may be employed by controller 60 of **FIG. 1** for prevention of vasovagal syncope. Upon detection of the sharp increase in contractility from one cardiac cycle to the next, the pacing rate is immediately increased from an initial pacing rate, PR1, (which may be a programmed base rate such as 60 bpm) to a syncope prevention rate, PR2 (which may be a programmable value, for example, 20, 30 or 40 bpm above the initial pacing rate, PR1). The increase from initial pacing rate, PR1, to syncope prevention rate, PR2, could be a linear increase performed during time T1 which may be, for example, in the range of three to five seconds. The heart is then continuously paced at syncope prevention rate, PR2, for time T2 which may be, for example, in the range of 30 seconds to 3 minutes. Then, the pacing rate is reduced from syncope prevention rate, PR2, to initial pacing rate, PR1, during T3 wherein T3 is, for example, in the range of 10 to 30 seconds. In the alternative, each individual time value T1, T2 and T3 is not separately programmable. Rather, the overall time period T4 is programmed, and the pacemaker automatically calculates the subportions T1, T2 and T3 in accordance with prestored timing ratios. For ease of implementation T1, T2, T3 and T4 could be replaced with the number of the corresponding beats.

[0044] By increasing the heart rate to syncope prevention rate, PR2, the drop in blood pressure that would otherwise occur during the onset of vasovagal syncope syndrome is at least partially compensated. Although the increase in the heart rate may not be sufficient to prevent the blood pressure from dropping to the point where the patient feels lightheaded or dizzy, it is typically sufficient to prevent the blood pressure from dropping to the point wherein the patient becomes unconscious. Even if the patient ultimately becomes unconsciousness, the loss of consciousness is delayed by at least some amount permitting the patient to take whatever action is necessary to ensure his or her safety. For example, if the patient is driving a vehicle, the patient, upon becoming lightheaded and dizzy, will preferably pull the vehicle to the side of the road. Thus, even if the patient loses consciousness, the delay in loss of consciousness may be sufficient to save the patient from injury.

[0045] By triggering the increase in pacing rate based upon a contractility measurement, the increase is triggered much more promptly than typically occurs within pacemakers configured to detect the onset of vasovagal syncope based upon bradycardia. As noted above, by the time a pacemaker has detected bradycardia, the blood patient of the patient may already have dropped to the point where a subsequent increase in the heart rate is ineffective for preventing immediate loss of consciousness. By triggering the increase in heart rate based upon a sharp change in contractility, the increase is typically triggered within one or two beats subsequent to the initial drop in blood pressure which marks the onset of vasovagal syncope. By significantly increasing the heart rate before the blood pressure has a chance to drop significantly, the pacemaker thereby either prevents any drop in blood pressure or, at the very least, defers the drop in blood pressure thereby delaying or preventing unconsciousness.

[0046] The steps performed by the components of **FIG. 1** for vasovagal detection and prevention are summarized in **FIG. 3**. Briefly, at step 200, device 10 inputs the electrical impedance signals, pressure, and/or accelerometer signals. At step 202, device 10 determines the contractility and, at step 204, compares the detected contractility with the average contractility. If, at step 205, the contractility increases to the point where the detected contractility minus the average contractility exceeds a predetermined threshold, then step 206 is performed wherein the device 10 immediately increases the pacing rate to the vasovagal syncope preventative pacing mode using the pacing rate defined in **FIG. 2**. Otherwise, processing returns to step 200 wherein device 10 continues to monitor the input impedance, pressure and accelerometer signals. Vasovagal syncope preventative pacing mode performed at step 206 is administered, as described in connection with **FIG. 2**, for example, for about 30 seconds to 3 minutes. Then, the heart rate is reduced to a base pacing rate or pacing is eliminated completely. Ultimately, processing returns to step 100 to monitor impedance, accelerometer signals and/or pressure.

[0047] Thus, **FIG. 3** illustrates, at a high level, steps

performed to predict and prevent vasovagal syncope using device 10. As described above, device 10 additionally performs numerous other functions simultaneously, such as monitoring the heart to detect heart arrhythmias, such as atrial tachycardia. If a significant heart arrhythmia is detected while the pacemaker is administering the vasovagal syncope preventative pacing mode, the preventative pacing is terminated and the pacemaker administers whatever therapy is appropriate for the arrhythmia. The therapy may include the administration of defibrillation pulses or the like.

[0048] **FIGS. 4 and 5** illustrate a sequence of steps performed to provide vasovagal preventative pacing within device 10 if the device is configured to pace at a sensor indicated rate (SIR). SIR pacing is described in further detail in U.S. Patent No. 5,476,483 to Bomzin et al., entitled "System and Method for Modulating the Base Rate During Sleep for a Rate-Responsive Cardiac Pacemaker", which is incorporated by reference herein.

[0049] The sequence of steps illustrated in **FIGS. 4 and 5** are generally in accordance with the techniques described above. Hence, the sequence of steps of **FIGS. 4 and 5** will not be described in detail but merely summarized herein. Steps 300, 302, 304, 314, 320, 322 and 312 are steps performed by device 10 in response to the initial detection of a sharp increase in contractility wherein the vasovagal preventative pacing mode is initiated. Here, the patient is paced at either the vasovagal preventative pacing rate (VPPR) illustrated in **FIG. 2** or a predetermined SIR, whichever is greater. If a further increase in contractility is detected while the patient is already within the vasovagal preventative pacing mode, the pacemaker continues to pace at either SIR or VPPR, whichever is greater. This is illustrated by steps 300, 302, 304, 314, 316, 322 and 312. Note that the vasovagal preventative pacing mode will be reinitiated so long as the contractility remains far above the average contractility level (step 304), i.e., the detected contractility minus the average contractility exceeds the predetermined threshold. In this regard, if the period for vasovagal preventative pacing has completed (step 216), VPPR pacing is suspended only as long as needed to detect that the contractility is still high and, during a subsequent execution of the steps of **FIG. 4**, VPPR pacing is again triggered.

[0050] Once contractility has dropped to an acceptable level, the pacemaker takes the patient out of the vasovagal preventative pacing mode. This is provided by steps 300, 302, 304, 306, 316, 308, 310 and 312 and also by steps 300, 302, 304, 308, 316, 308, 318 and 312.

[0051] What have been described are techniques for predicting and preventing vasovagal syncope within patients subject to recurrent vasovagal syncope using an implanted cardiac pacing device. Principles of the invention are applicable to predicting and preventing other types of syncope as well such as other types of neurocardiogenic syncope. Other physiological sensor that reflect the contractility change of the heart can be employed, such as ventricular gradient (also known as paced depolarization interval (PDI); pre-ejection period (PEP), etc.). The techniques are also applicable for use with other implantable medical devices, such as ICDs or the like. Thus, the specific examples herein are intended merely to be illustrative of the invention and should not be construed as limiting the scope of the invention.

Claims

1. A system in an implantable device for detecting the possible onset of vasovagal syncope within a patient, the system comprising:

   an input circuit for receiving a signal representative of the contractility of the heart of the patient; and

   a controller for determining, based on the detected contractility, whether there is a significant risk of the onset of vasovagal syncope.

2. The system of Claim 1, wherein the input circuit receives a signal representative of at least one of the contractility of the ventricles of the heart of the patient, electrical blood impedance, heart wall acceleration or blood pressure within the left ventricle.

3. The system of Claim 1, wherein the controller determines an average contractility value of the heart, stores the average contractility value, compares a current value for the contractility of the heart to the average contractility of the heart stored in the memory register, and determines whether there is a significant risk of the onset of vasovagal syncope based upon the comparison of the current value of the contractility to the average value.

4. The system of Claim 1, further including a pulse generator and wherein the controller, upon detecting a significant risk of the onset of vasovagal syncope, controls the pulse generator to generate pacing pulses for applying to the heart so as to reduce the likelihood of the vasovagal syncope.

5. The system of Claim 4, wherein the controller:

   incrementally increases a pacing rate from a preexisting pacing rate to a predetermined vasovagal syncope prevention rate during a first predetermined on number of beats;

   maintains the pacing rate at the predetermined vasovagal syncope prevention rate during a second predetermined on number of beats; and

   incrementally decreases the pacing rate from the vasovagal prevention rate to the preexisting pacing rate during a third predetermined on

number of beats.

6. The system of Claim 5, wherein the first, second and third predetermined period of times are in the range of 3 to 5 seconds, 30 to 180 seconds, and 10 to 30 seconds, respectively.

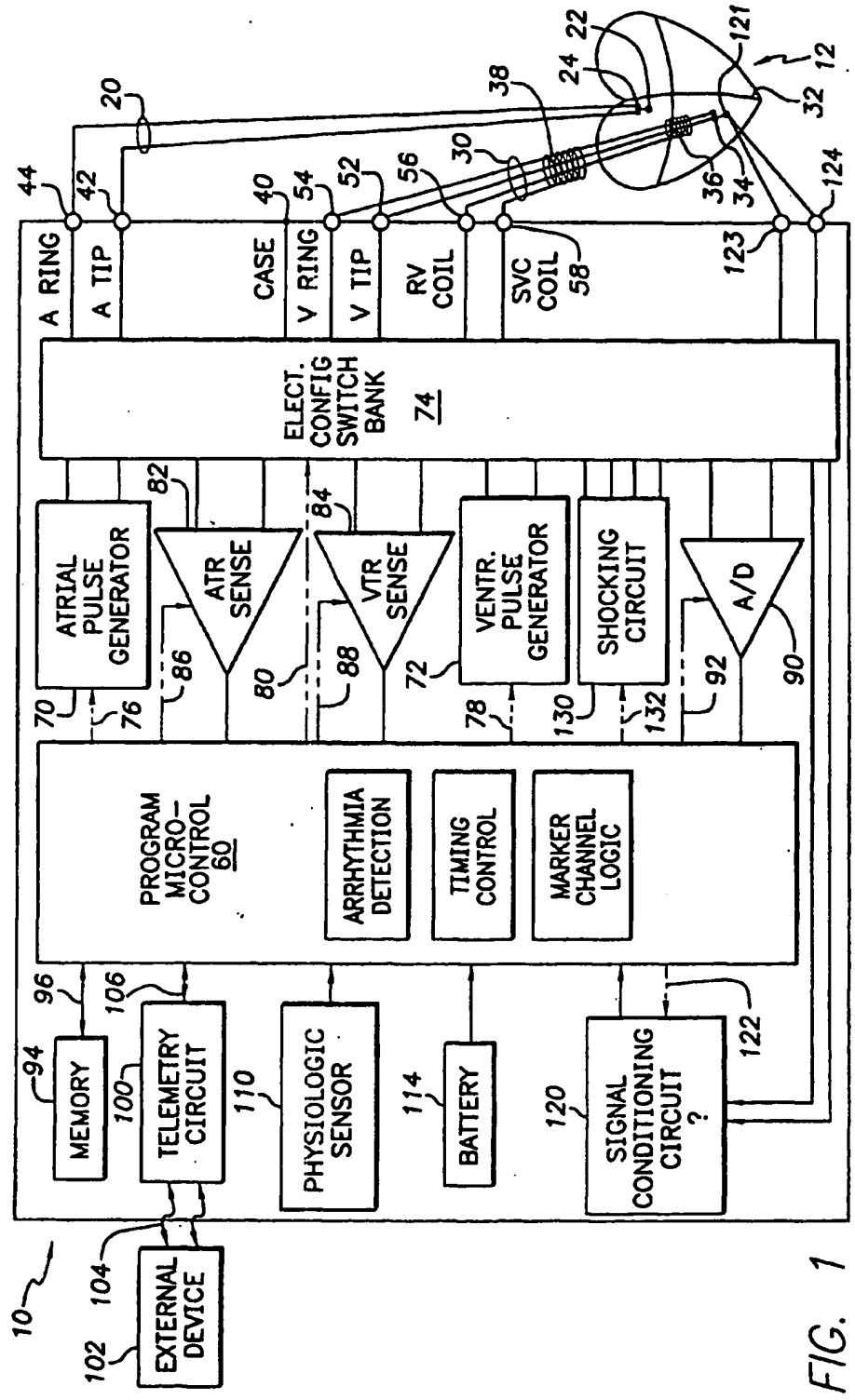
7. The system of Claim 5, wherein the vasovagal syncope prevention rate is in the range of 20 to 40 beats per minute above the preexisting pacing rate.

8. The system of Claim 5, wherein the preexisting pacing rate is a predetermined base pacing rate.

9. The system of Claim 5, wherein the preexisting pacing rate is a sensor responsive pacing rate.

10. A method for use in an implantable device for detecting the possible onset of vasovagal syncope within a patient comprising the steps of:

    detecting the contractility of the heart; and
    determining, based on the detected contractility, whether there is a significant risk of the onset of vasovagal syncope.


A RING
A TIP
CASE
V RING
V TIP
RV COIL
SVC COIL
58
ELECT. CONFIG SWITCH BANK
74
ATRIAL PULSE GENERATOR
ATR SENSE
VTR SENSE
VENTR. PULSE GENERATOR
SHOCKING CIRCUIT
A/D
PROGRAM MICRO-CONTROL 60
ARRHYTHMIA DETECTION
TIMING CONTROL
MARKER CHANNEL LOGIC
MEMORY
TELEMETRY CIRCUIT
PHYSIOLOGIC SENSOR
BATTERY
SIGNAL CONDITIONING CIRCUIT ?
EXTERNAL DEVICE
10
12
20
22
24
30
32
34
36
38
40
42
44
52
54
56
70
72
76
78
80
82
84
86
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90
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102
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110
114
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121
122
123
124
130
132

FIG. 1

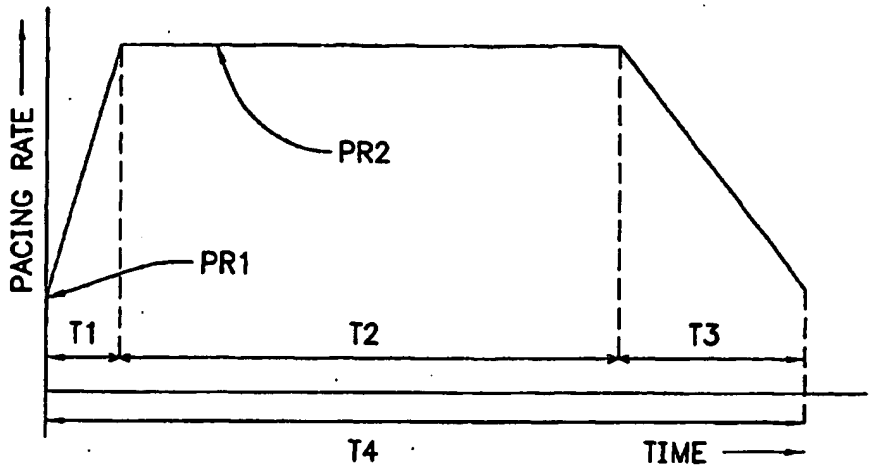
PACING RATE
PR2
PR1
T1
T2
T3
T4
TIME

FIG. 2

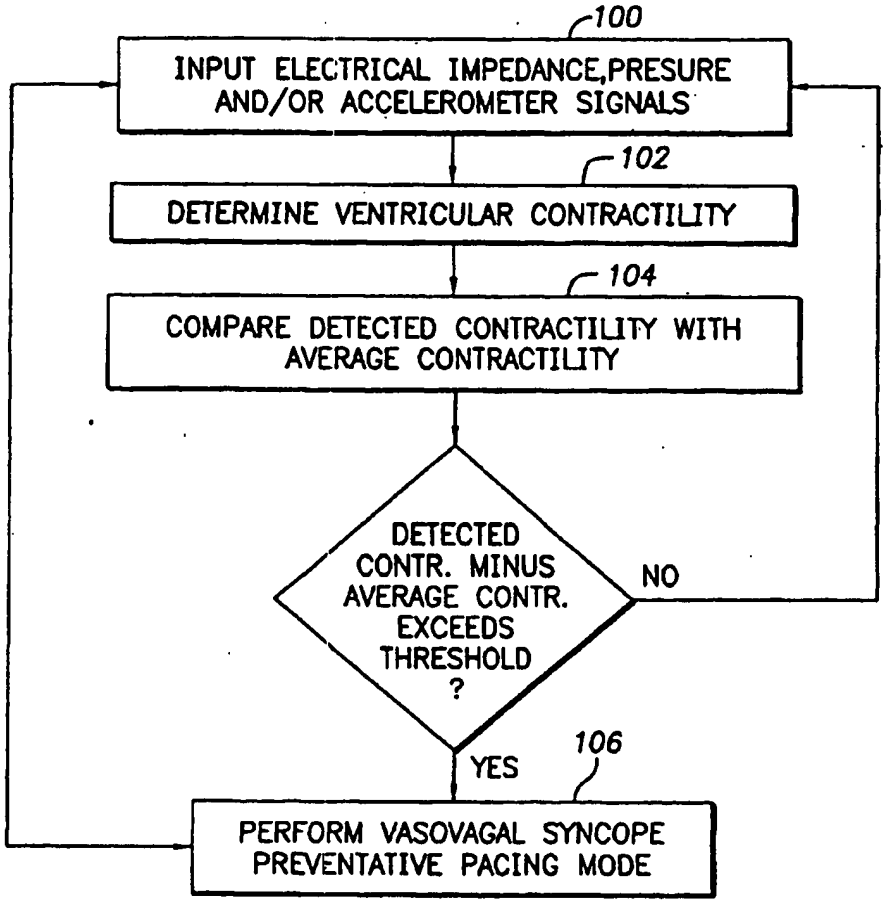
100
INPUT ELECTRICAL IMPEDANCE,PRESURE AND/OR ACCELEROMETER SIGNALS
102
DETERMINE VENTRICULAR CONTRACTILITY
104
COMPARE DETECTED CONTRACTILITY WITH AVERAGE CONTRACTILITY
DETECTED CONTR. MINUS AVERAGE CONTR. EXCEEDS THRESHOLD ?
NO
YES
106
PERFORM VASOVAGAL SYNCOPE PREVENTATIVE PACING MODE

FIG. 3

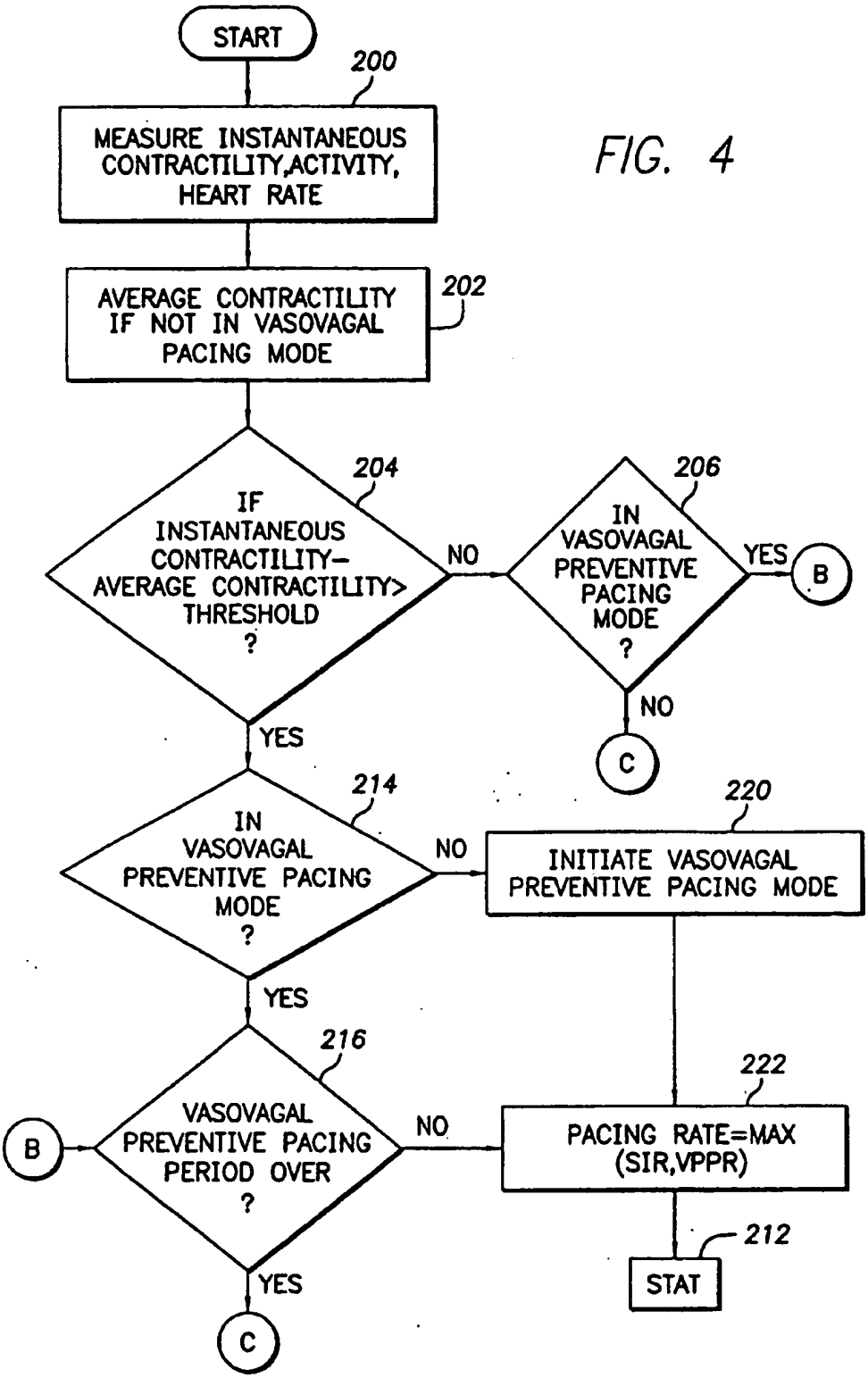
START
200
MEASURE INSTANTANEOUS CONTRACTILITY,ACTIVITY, HEART RATE
AVERAGE CONTRACTILITY IF NOT IN VASOVAGAL PACING MODE
202
204
IF INSTANTANEOUS CONTRACTILITY- AVERAGE CONTRACTILITY> THRESHOLD ?
NO
206
IN VASOVAGAL PREVENTIVE PACING MODE ?
YES
B
NO
C
YES
214
IN VASOVAGAL PREVENTIVE PACING MODE ?
NO
220
INITIATE VASOVAGAL PREVENTIVE PACING MODE
YES
216
VASOVAGAL PREVENTIVE PACING PERIOD OVER ?
B
NO
222
PACING RATE=MAX (SIR,VPPR)
212
STAT
YES
C

FIG. 4

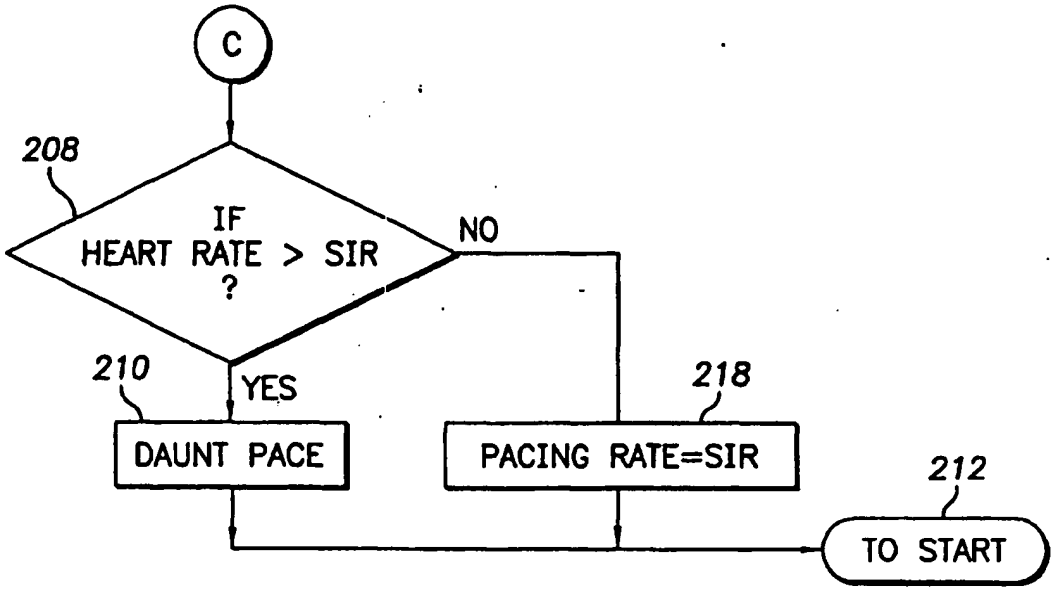
C
208
IF
HEART RATE > SIR
?
NO
210
YES
218
DAUNT PACE
PACING RATE=SIR
212
TO START

FIG. 5